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SOME PARAMETERS OF HEART RATE CHANGE: PERCEPTUAL VERSUS MOTOR TASK REQUIREMENTS, NOXIOUSNESS, AND UNCERTAINTY

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ABSTRACT

Sensory-motor integration and physiological patterns were investigated in a modified reaction time task. Following a READY signal, one of 2 DISCRIMINATIVE signals indicated that a right or left reaction was to be made to a GO signal. For one group, the DISCRIMINATIVE and GO signals occurred simultaneously; for another group, the GO signal was delayed 10 sec. In different sessions, shock occurred with the DISCRIMINATIVE signal on 0%, 33%, or 100% of the trials. The basic pattern of heart rate response was the same in all conditions, namely, acceleration followed by deceleration immediately prior to the DISCRIMINATIVE and GO signals. All experimental manipulations appeared to contribute to cardiac deceleration; for example, the greatest decrease occurred prior to the simultaneous DISCRIMINATIVE-GO signal with 33% shock probability. The least deceleration (and fastest reaction times) occurred to the delayed GO signal. Anticipation of a motor response and or shock also accentuated the accelerative limb of the heart rate curve, as well as producing increased skin conductance. Muscle action potentials from the chin showed an equivocal relationship to cardiac acceleration (or less deceleration) and to faster reaction times. Results are discussed in terms of an attentional hypothesis, and their relevance to speculations by Lacey and Obrist is examined.

DESCRIPTORS: Heart rate, Sensory-motor integration, Noxiousness, Uncertainty, Attention. (J. R. Jennings)

There has been burgeoning interest in the fact that heart rate sometimes accelerates and sometimes decelerates in response to environmental demands (e.g., Lacey, Kagan, Lacey, & Moss, 1963; Coquery & Lacey, 1966; Lacey, 1967; Obrist, Webb, Sutterer, & Howard, 1970a, 1970b). The understanding of any functional relationship between the direction of heart rate change and the organism's behavioral reaction to environmental demands requires the identification

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of relevant situational parameters. The present experiment was designed to investigate the influence of (a) perceptual versus motor task requirements, (b) noxiousness, and (c) uncertainty. Additional analyses were made of (d) the coupling between somatic (striate musculature) and cardiac activity, and (e) the relationship of physiological changes to reaction time.

Obviously, no single experiment could settle all the complex problems implicit in the five topics just noted. Nevertheless, it is important to manipulate within a single experiment the relevant parameters so that their interactions may be observed and issues sharpened.

METHOD

Overview of Experimental Design

Perceptual versus motor requirements were manipulated by establishing two variations of a reaction time task. In the first, a READY signal was followed in 10 sec by one of two lights (the DISCRIMINATIVE signal) indicating that a right or left response was to be made. Some subjects experienced simultaneously a reaction (GO) signal which immediately initiated the response; for other subjects the GO signal, and hence the response, was delayed for another 10 sec. In this way a continuum of perceptual to motor requirements was created. In the delayed reaction conditions, response to the DISCRIMINATIVE signal involved no overt motor act, but only a perceptual discrimination. In the no delay condition, involving simultaneous DISCRIMINATIVE and GO signals, responses included perceptual and motor components. Finally, subjects in the delayed reaction condition were confronted with a simple reaction time task when the GO signal appeared. In sum, the arrangement made possible an analysis of the extent of contributions to heart rate deceleration of (1) a task which was chiefly perceptual, (2) a task which combined both perceptual and motor components, and (3) a task which was chiefly motor.

The influence of noxiousness and uncertainty was investigated by introducing shock on either 100%, 33%, or 0% of the trials. In the 100% shock treatment, *S*s received a 1 sec shock with the DISCRIMINATIVE signal. In the 33% treatment, shock was associated with the DISCRIMINATIVE signal on a randomly selected 33% of the trials. In the 0% treatment, *S*s did not receive any shock. A repeated measurements design was used for the shock manipulation; each *S* received all shock treatments, one treatment per session. The three sessions consisted of 60 trials each and were on separate days within a single week.

The basic experimental design is illustrated in Table 1. Its major feature was the use of two identical latin squares (see Winer, 1962, p. 553) for the immediate and delayed reaction conditions. Three probabilities of shock, counterbalanced over sessions, formed the within square treatments.

Subjects

Thirty male subjects (*S*s), 18 to 28 years of age, were obtained through the student placement bureau. The immediate and delayed reaction conditions each contained three groups of 5 *S*s each, assigned on a random basis.

TABLE 1

Outline of experimental design

Group	Condition		
	Session I	Session II	Session III
Immediate Reaction			
Group 1	0% Shock	33% Shock	100% Shock
Group 2	33% Shock	100% Shock	0% Shock
Group 3	100% Shock	0% Shock	33% Shock
Delayed Reaction			
Group 4	0% Shock	33% Shock	100% Shock
Group 5	33% Shock	100% Shock	0% Shock
Group 6	100% Shock	0% Shock	33% Shock

After reporting to the laboratory, *S*s were informed that they would receive a moderately painful shock and were allowed to withdraw at this or any other point in the experiment. None withdrew for this reason, but 2 *S*s failed to return for all the sessions and the data for 2 were discarded due to equipment malfunction. These *S*s were replaced through the placement bureau.

Procedure

*S*s sat in a semi-reclining chair facing a display of signal lights mounted in a diamond configuration. Two red lights on the right and left sides of the display served as DISCRIMINATIVE signals. A yellow READY signal was centered below the DISCRIMINATIVE signals, and a green GO signal was centered above. Each signal light when lit remained on for 1 sec. Two darkroom timers indicated the elapsed time between signals. These were located in the bottom right and left corners of the display, in easy view of the *S*.

The READY signal marked the beginning of a trial. Simultaneously with its onset, one of the darkroom timers started. After 10 sec, either the left or right DISCRIMINATIVE signal came on. For *S*s in the immediate reaction condition the GO signal occurred with the DISCRIMINATIVE signal. For *S*s in the delayed reaction condition a further 10 sec delay—indicated on the second darkroom timer—was interposed before the GO signal. All *S*s responded to the GO signal by pushing a reaction key to the right or left depending upon which DISCRIMINATIVE signal had lit. The sequence of right and left DISCRIMINATIVE signals was randomized.

The reaction key consisted of a double throw telephone switch placed on the left arm of the chair. *S*s were told to keep the left hand in contact with the key throughout the trials and to respond as quickly as possible. They were also told to pay attention to the darkroom timers in order to be sure when the delay intervals would end. Reaction time was measured with a Standard Timer.

There were 60 trials per session, separated by random intertrial intervals with a mean of 25 sec and a range of 12 to 32 sec. Five catch trials, on which the GO signal did not appear, were included randomly within the last 50 trials of each session.

Each shock treatment was explained prior to the session on which it was to be given. It was emphasized that the purpose of the shock was to study changes in physiological responding when attention was directed toward a stimulus situation with noxious components. *S*s were clearly told that shock was in no way contingent upon responses. This was particularly emphasized for the 33 % shock treatment.

Prior to the experimental trials of the first session, each *S* was asked to adjust the intensity of his own shock. Shocks were presented in an ascending series and the intensity judged moderately painful was used throughout the rest of the experiment for that *S*. The mean intensity selected was 2.5 ma. The range was from .5 ma to 5.5 ma.

Respiration was controlled by asking *S*s to breathe to a metronome beat. Before each session, each *S* set the metronome to a rate that allowed him to breathe comfortably and then practiced breathing to the metronome for about 10 min.

Apparatus and Electrode Placement. Heart rate (HR), skin resistance (SR), muscle potential (EMG), respiration, and stimulus events were recorded on a Beckman Type R Dynograph located in an adjacent room. HR was recorded from chest electrodes using a Beckman cardiotachometer. SR was recorded from the thenar and hypothenar surfaces of the right palm using Beckman Ag-AgCl electrodes, 1 cm in diameter, and a cornstarch paste (Edelberg, 1967). A constant current (10 μamp) modified Wheatstone bridge was employed. EMG was recorded from the midpoint of the frontal surface of the chin and a point about 1 inch below the tip of the chin. Respiration was monitored from a strain gauge on a belt located just below the rib cage.

A concentric, aluminum shock electrode (Tursky, Watson, & O'Connell, 1965) was placed on the dorsal surface of the left forearm following preparation of the skin to lower resistance. A ground made from copper sheet metal was placed around the arm above the electrode in order to reduce electrical interference during shock.

Data Reduction

Only data from the first 15 and last 15 trials were quantified. This reduced the amount of data to be read, and still allowed an analysis of temporal changes in response.

Heart Rate was read beat-by-beat starting 10 beats prior to the READY signal. The data were then arranged so that there were always 12 beats within each 10-sec anticipation interval.[1] Six of the 12 beats were those immediately following one signal and 6 were those immediately preceding the next signal. This resulted in some readings in the middle of the interval being dropped for *S*s with an average HR above 72 bpm. For *S*s with a lower average HR, there was repetition of some beats in the middle of the 10-sec intervals. The 10 beats following the GO signal were also read.

Skin Resistance was read as the minimum resistance in successive 5 sec intervals starting 5 sec prior to the READY signal and terminating 5 sec after the GO

[1] This arrangement was suggested by J. Lacey, personal communication, 1969.

signal. There were thus two readings within each anticipation period. Resistance measures were converted to skin conductance (SC) before further analysis.

Muscle Potentials were classified as occurring or not occurring in the successive 5 sec intervals described for skin conductance. Occurrence of EMG was noted if a burst exceeded 50 microvolts and had the characteristic form of an EMG burst.

Respiration was assessed by measuring the time between successive inspiration peaks. This was done for the 7 breaths following the READY signal, thus encompassing the entire trial.

RESULTS

The Beat by Beat Cardiac Reactions

The pattern of HR change was similar in all experimental treatments. After the READY signal, a slight deceleration tended to occur. In both immediate and delayed reaction conditions, there followed a very evident acceleratory phase, which again changed to deceleration just prior to the DISCRIMINATIVE (or DISCRIMINATIVE-GO) signal. In the delayed reaction condition, a similar response was evident in the pre-GO interval. Fig. 1 presents the mean HR curves of the delayed reaction condition for the 100% shock treatment. The mean response values for the first and last 15 trials are plotted separately in order to illustrate within session adaptation. Fig. 2 presents the mean HR curves of the immediate reaction condition for the three shock treatments plotted separately.

Analysis of variance was used to assess the reliability of the cardiac response pattern within each of the treatment combinations. The data were separated into 12 blocks, one for each combination of shock probability (3 levels), immediate or delayed reaction (2 levels), and first or last 15 trials within a session (2 levels). For example, one block contained the data for the first 15 trials of

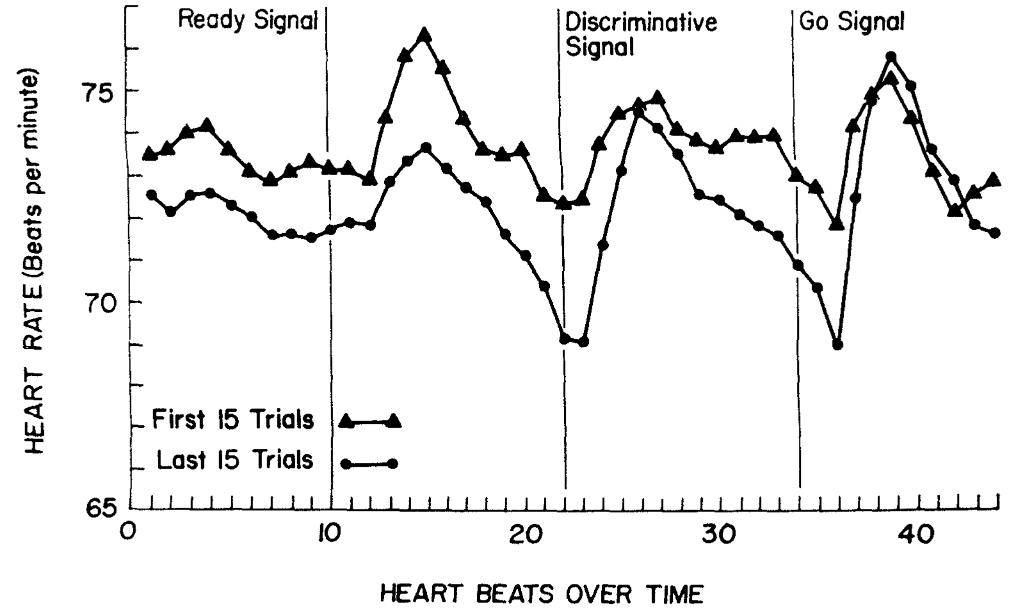

FIG. 1. Mean beat-by-beat heart rate, starting 10 beats prior to the READY signal, for delayed reaction group, 100% shock probability. Shock was delivered simultaneously with the DISCRIMINATIVE signal.

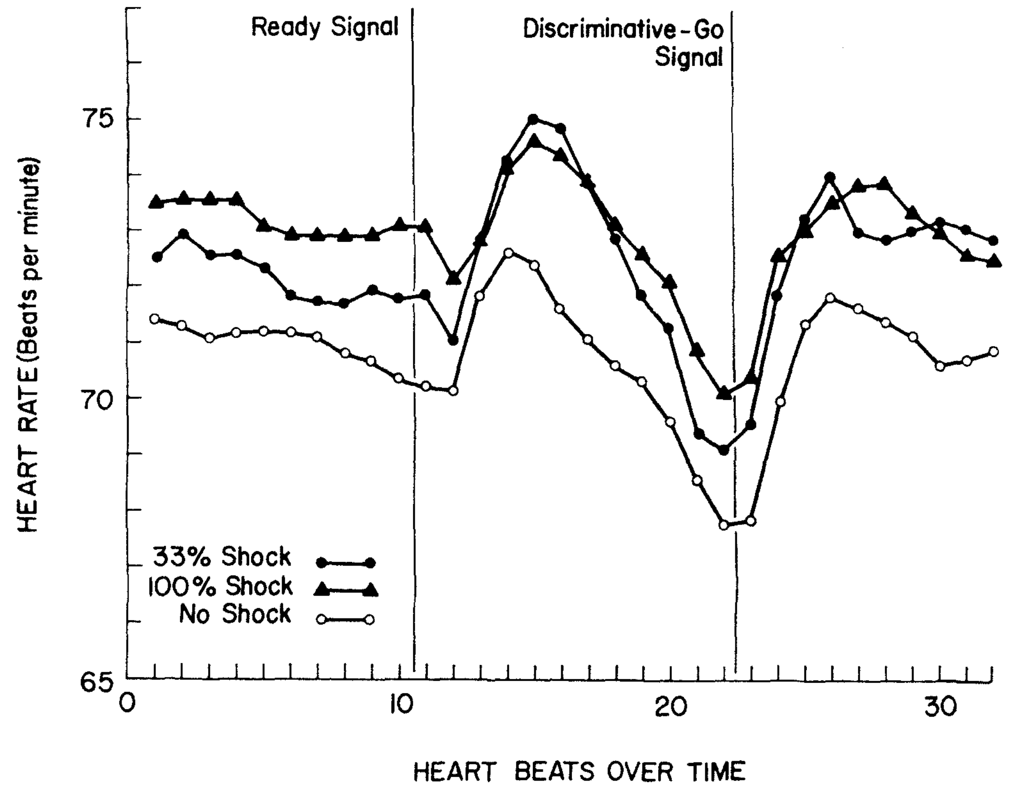

FIG. 2. Mean beat-by-beat heart rate, starting 10 beats prior to the READY signal, for 0%, 33%, and 100% shock probabilities, immediate reaction group. Shock was delivered simultaneously with the DISCRIMINATIVE-GO signal.

the immediate reaction, 0% shock treatment. The data for each block was then subjected to an analysis of variance with subjects as one factor, individual heart beats over time as a second factor, and trials (15) as a nested third factor. (Wilson, 1967, has described fully this type of analysis as well as the general statistical approach.)

In every analysis, statistically significant ($p < .005$) effects were found for subjects, beat-by-beat HR, and the interaction of subjects × HR. (Significance levels were based on one half the actual degrees of freedom in order to adjust for the nonindependence of heart beats—Wilson, 1967.) This means that the general response patterns depicted in Figs. 1 and 2 are highly reliable.

An examination of Fig. 1 suggests that although the HR pattern is similar for the first and last 15 trials of a session, differences do exist in the accentuation of certain features of the response as well as in the average HR level. To test these possibilities, two mean HR curves were obtained for each *S* by averaging his first and last 15 trials. Six analyses of variance similar to those described above were then performed, one for each combination of the three shock probabilities and immediate versus delayed reaction. For the delayed condition, the progressive accentuation of the response curve (primarily the decelerative limb) was reflected in significant trial (first vs last 15) × HR interactions. The effect was strongest at the 100% shock level ($F = 2.09$, actual $df = 43, 602$, adjusted $p < .005$); was intermediate with no shock ($F = 1.75$, adjusted $p < .05$); and the 33% shock group showed a similar trend ($F = 1.55$, adjusted $p < .10$). The trial × HR interaction was not significant in any of the immediate reaction group analyses.

The decrease in overall HR level from the first to last 15 trial period was evident in three of the six treatment combinations. Two of the three cases were statistically significant (immediate reaction, 100% shock; delayed reaction, 33% shock; both $p < .05$) and a third was of border line significance (immediate reaction, 33% shock; $p < .10$).

Derivation of Response Scores

For the next step in the analysis, response scores were derived which summarize the physiological changes occurring in each anticipation interval. These scores were used as dependent variables in a set of latin square and other analyses based on the design shown in Table 1. Note that three squares were actually created by this design, as the delayed reaction condition contained anticipation intervals to both the DISCRIMINATIVE and GO signals.

Heart Rate. Each *S*'s HR responses were averaged within sessions (with the first and last 15 trials combined) and the following measures were derived: The initial (BASE) level was defined as the mean of the 10 beats prior to the READY signal. Three measures were used to describe the biphasic response pattern between the READY and DISCRIMINATIVE signal (see Figs. 1 and 2): (a) maximum minus base (MAX-BASE) was the maximum HR during the 6 beats following the READY signal minus the initial level; (b) minimum minus base (MIN-BASE) was the minimum HR during the 4 beats preceding the DISCRIMINATIVE signal minus initial level; and (c) the difference between the maximum and the minimum (MAX-MIN). A comparable set of scores was used to describe the response preceding the GO signal in the delayed condition.

These scores were not corrected for initial levels as the BASE to response score correlations were generally quite low (mean correlation = .01). A latin square analysis (Table 1) was performed using the BASE scores as the dependent variable in order to assess the effect of the manipulations on initial levels. The only significant effect was due to higher base rates during the third experimental session ($F = 3.41$, $df = 2, 48$, $p < .05$). This difference in initial level was also reflected in the analysis on the MAX-BASE scores from the interval prior to the DISCRIMINATIVE signal, in which the sessions effect was significant. In general, the balance of the design should negate any effect due to session upon the other experimental variables.

Skin Conductance. A single score was used to assess changes in SC, namely, the maximum SC in the 5 sec interval preceding the DISCRIMINATIVE signal minus the maximum skin conductance in the 5 sec interval preceding the READY signal. SC reactions prior to the GO signal were strongly influenced by shock accompanying the preceding DISCRIMINATIVE signal and, therefore, are not discussed. As in the case of HR, the correlations between SC base and response scores were quite low (mean $r = .08$), and so no corrections for initial levels were made.

Muscle Potential. Occurrence or nonoccurrence of EMG activity within each half of an anticipatory interval was coded as a 1 or 0, respectively. The association between a given pattern of 1's and 0's and another variable, such as HR, was then tested.

Respiration. No response scores were derived from the respiration measures previously described. Descriptive statistics (means, variances, and frequency distributions) were computed in order to assess whether *S*s had controlled their respiration as requested. These statistics showed that the *S*'s breathing was in general regular and free of gross abnormalities, but that the respiration training had not been entirely successful. Frequency tabulations of all the respiratory peaks within each of the six shock-delay treatment combinations showed a significant increase in the number of peaks during the first 5 sec after the READY signal and during the first 5 sec after the DISCRIMINATIVE signal. These departures from a random frequency distribution were significant beyond the .01 level using a Kolmogorov-Smiroff test. Similar tests on the distribution of respiratory peaks across the experimental conditions within the 5 sec intervals showed no differences among the conditions. Thus, although the general cardiac deceleratory effect within an experimental trial may have been influenced by respiration, it is unlikely that respiratory changes could explain any differences found between experimental conditions. Moreover, recent work (e.g., Headrick & Graham, 1969) has questioned whether respiration influences heart rate under conditions similar to those used in this experiment.

The effects of experimental manipulations on the above response scores are described below. Results are organized around the five issues mentioned in the introduction: perceptual versus motor task requirements, noxiousness, uncertainty, the coupling between somatic and cardiac activity, and the relationship of physiological changes to reaction time.

Perceptual versus Motor Task Requirements

Heart Rate. This issue was studied using the three latin squares described earlier. Reactions prior to the DISCRIMINATIVE-GO signal (immediate reaction condition) were compared, first, with reactions to the DISCRIMINATIVE signal (delayed condition) and, second, with reactions to the GO signal (also delayed condition). Then, reactions to the DISCRIMINATIVE and GO signals were compared within the delayed condition. The dependent variables were the MAX-BASE, MIN-BASE, and MAX-MIN scores, which measured the accelerative, decelerative, and peak-to-trough characteristics of the biphasic response curve.

Deceleration tended to be greater prior to the DISCRIMINATIVE-GO signal of the immediate reaction condition than prior to the delayed GO signal. (The MIN-BASE scores were -3.99 as opposed to -2.47; $F = 3.18$, $df = 1, 24$, $p < .10$. An effect of similar magnitude was observed for the MAX-MIN scores.) On the other hand, a comparison between the DISCRIMINATIVE-GO and the DISCRIMINATIVE signals failed to show any differences.

The final comparison was between anticipatory reactions within the delayed condition. For statistical reasons, this comparison was made using a repeated measures design ignoring the sessions and order effects. Deceleration prior to the DISCRIMINATIVE signal was more pronounced than that before the GO signal ($F_{\text{MIN-BASE}} = 6.29$, $df = 1, 14$, $p < .05$). An interaction between shock probability and anticipatory interval (pre-DISCRIMINATIVE vs pre-GO) was also significant ($F = 5.62$, $df = 2, 28$, $p < .01$). The difference between the MIN-BASE scores for the two signals was .07 beats in the 0% probability condition, −1.89 in the 33% shock condition, and −1.52 in the 100% shock conditions.

Skin Conductance, Muscle Potentials, and Respiration. A latin square analysis of SC changes preceding the DISCRIMINATIVE-GO and DISCRIMINATIVE signals did not yield any significant results as a function of delayed versus immediate reaction. However, as will be discussed below, this negative finding requires some qualification. The effect of experimental manipulations on patterns of EMG activity was tested by a series of Friedman nonparametric analyses of variance; no significant results were obtained for delayed versus immediate reaction, or for any of the other experimental variables. As already described, respiration was not affected by the delay manipulation.

In brief, both perceptual and motor demands produced anticipatory cardiac deceleration. Greatest deceleration occurred prior to the signal which placed the greatest perceptual demands on the subject (DISCRIMINATIVE-GO), but which also involved an anticipated motor response. The least deceleration occurred prior to the simple GO signal. Moreover, it appears that the expectation of shock also contributed to cardiac deceleration, at least prior to the DISCRIMINATIVE signal of the delayed group. No significant effects of differing perceptual-motor demands on cardiac acceleration were obtained from the present analyses. However, as in the case of skin conductance, this conclusion will have to be modified in the light of analyses to be discussed subsequently.

Noxiousness and Uncertainty

Heart Rate. The analysis of the DISCRIMINATIVE and DISCRIMINATIVE-GO signal squares showed a significant effect of shock probability on HR. Both the MAX-BASE ($F = 3.80$, $df = 2, 48$, $p < .05$) and the MAX-MIN ($F = 5.49$, $p < .01$) scores showed reliable differences. The largest MAX-MIN change (mean = 7.72) occurred in the 33% shock treatment, followed by the 100% shock (mean = 6.08) and the no shock (mean = 5.34). Tukey's *b* test showed significant differences not only between the 0% and 33% shock ($p < .01$), but also between 33% shock and 100% shock ($p < .05$). The analogous results for the MAX-BASE means showed the 0% and 33% shock levels to differ at the .05 probability level, while differences between the 33% and 100% shock treatments were marginally significant ($p < .10$). The fact that the MAX-MIN scores yielded higher significance levels than the MAX-BASE scores might indicate that shock probability was influencing cardiac deceleration as well as acceleration. This indication is supported by the previously described significant interaction between shock probability and type of signal anticipated. It will be recalled that for the delayed condition deceleration prior to the DISCRIMINATIVE signal was greatest for 33% shock probability and next for 100% probability. With no shock anticipated, deceleration was approximately equal prior to the DISCRIMINATIVE and GO signals.

Skin Conductance. Analyses showed consistently greater SC increases with 100% and 33% shock probability. Thus, the latin square comparison between the anticipatory reactions to the DISCRIMINATIVE-GO and DISCRIMINA-

TIVE signals were highly significant for the shock manipulation ($F = 6.89$, $df = 2, 48$, $p < .005$). Tukey *b* tests showed that the treatment means for the 100% and 33% shock were significantly different from the no shock mean ($p < .05$), but not from each other. The mean changes in conductances (μmhos) prior to the DISCRIMINATIVE-GO signal were: 0% = .262, 33% = .410, 100% = .496. The corresponding means for the DISCRIMINATIVE signal were: 0% = .035, 33% = .458, 100% = .445.

Muscle Potential and Respiration. As previously noted, neither of these variables showed statistically significant differences due to shock probability.

To summarize, both the accelerative and decelerative limbs of the HR response were influenced by the shock manipulation. Noxiousness per se (100% vs 0%) increased both acceleration and deceleration, but not so much as noxiousness and uncertainty combined (33%). Indeed, uncertainty itself (33% vs 100%) had a significant effect, either in its own right or by enhancing noxiousness. The former possibility seems more likely since shock also resulted in anticipatory SC increases, while uncertainty did not add to this effect.

Coupling Between Somatic and Cardiac Activity

Each anticipatory interval was divided into two 5 sec periods and the following patterns of EMG activity identified: a 0/0 pattern indicated that no EMG burst occurred in either 5 sec period; a 0/1 pattern indicated that there was no EMG activity in the initial 5 sec of the anticipatory interval, but that one or more bursts did occur in the 5 sec immediately preceding the signal; a 1/0 indicated a pattern opposite to the preceding, namely, EMG occurrence followed by nonoccurrence; finally, a 1/1 pattern indicated EMG activity in both halves of the anticipation interval. A set of Friedman nonparametric analyses of variance indicated the occurrence of these patterns was not influenced by the experimental manipulations.

In order to study the relationship between EMG patterns and cardiac activity, each *S*'s HR scores were combined for trials showing the same pattern of EMG activity. For example, if an *S* showed 14 trials with a 1/0 pattern, the mean HR score for these 14 trials was computed. Similar means were computed for trials showing the 0/0, 0/1, and 1/1 patterns. Differences in HR responses corresponding to the different EMG patterns were then analyzed using *t* tests for dependent means. The degrees of freedom for the *t* tests varied from 4 to 14. Low degrees of freedom were associated with the tests involving the 1/1 pattern, as the majority of *S*s failed to show EMG activity during both halves of any given anticipation interval.

The results of the above analyses for shock treatments combined are contained in Table 2. That is, the results are reported only by the different types of anticipation intervals—the pre-DISCRIMINATIVE-GO, pre-DISCRIMINATIVE, and pre-GO intervals. The numbers presented are the mean HR change scores associated with the EMG patterns in the left column. Positive MAX-BASE scores indicate acceleration; negative MIN-BASE scores indicate deceleration. Tests of significance were performed on the differences between the HR scores associated with the two EMG patterns on the left; however, the actual

TABLE 2

Mean heart rate response scores on trials associated with different patterns of EMG activity

EMG Activity	Mean HR Change Scores					
	Immediate Reaction Group DISCRIMINATIVE-GO Signal		Delayed Reaction Group DISCRIMINATIVE Signal		Delayed Reaction Group GO Signal	
	MAX-BASE	MIN-BASE	MAX-BASE	MIN-BASE	MAX-BASE	MIN-BASE
0/1 versus 0/0	7.54 vs 4.91*	−3.33 vs −6.50***	5.98 vs 5.22	−3.13 vs −6.41*	7.38 vs 4.85**	−.06 vs −6.11***
1/1 versus 1/0	9.82 vs 5.95**	−5.87 vs −8.58*	8.71 vs 6.80	−5.42 vs −7.72	4.91 vs 9.43***	−4.42 vs −8.55
0/1 versus 1/0	7.49 vs 6.12	−3.20 vs −8.75*	5.98 vs 7.02	−3.13 vs −8.19**	7.72 vs 8.31	.02 vs −6.68**
1/1 versus 0/0	9.82 vs 4.51***	−5.87 vs −6.30	8.71 vs 5.85*	−5.42 vs −7.21	4.91 vs 4.58	−4.42 vs −8.57*
1/0 versus 0/0	6.54 vs 4.86*	−7.86 vs −5.81	7.30 vs 5.40	−8.11 vs −6.45	7.82 vs 4.99**	−6.57 vs −6.08
1/1 versus 0/1	9.45 vs 7.29	−6.28 vs −3.84	8.71 vs 4.97*	−5.42 vs −4.36	4.91 vs 5.99	−4.42 vs −1.21

Note.—Probability values are those for the significance of the difference between HR scores associated with the two EMG patterns. The significance was evaluated using t tests for dependent means, with degrees of freedom varying from 4 to 14.

* $p < .10$.

** $p < .05$.

*** $p < .01$.

difference scores are not presented in Table 2. For example, consider the first cell of Table 2. The mean HR acceleration associated with the 0/1 pattern was 7.54, while that associated with the 0/0 pattern was 4.91. The difference between these two (2.63) was significant at the .10 level, meaning that greater acceleration occurred on trials where a 0/1 pattern of EMG was observed. On the other hand, the MIN-BASE scores for 0/1 vs 0/0 indicate that significantly greater deceleration occurred on 0/0 EMG trials.

At first glance, the results presented in Table 2 do not seem to allow easy summary; on closer inspection, however, a consistent pattern does emerge. First, consider the MIN-BASE comparisons in the first four rows. These contrast trials where EMG activity occurred within 5 sec *immediately preceding* a signal with trials during which no such activity was observed. Of the 12 comparisons, all show less cardiac deceleration in the presence of EMG activity; 8 of these comparisons are statistically significant at the .10 level of confidence. Next consider the MAX-BASE comparisons in the last four rows (3 through 6). These contrast trials where EMG activity occurred in the *initial half* of an anticipation interval with trials where no such activity was observed. Nine of the 12 comparisons indicate greater cardiac acceleration in the presence of EMG activity. Five of these comparisons are significant at the .10 level; none of the 3 comparisons which point in the opposite direction are significant. It thus appears that the presence of somatic activity was associated with a mitigation of the decelerative limb of the HR response and an accentuation of prior acceleration. (It should be noted, however, that this conclusion does not take into consideration comparisons of the MAX-BASE differences in rows 1 and 2 or the MIN-BASE differences in rows 5 and 6. Although some of these comparisons were statistically significant, the interpretation of the EMG patterns being contrasted is ambiguous.)

Relationship of Physiological Changes to Reaction Time

Heart Rate and Reaction Time—Latin Square Analyses. Two significant effects emerged from latin square analyses of reaction time: first, immediate vs delayed reaction conditions ($F = 12.93$, $df = 1, 24$, $p < .005$); and second, session of the experiment ($F = 4.80$, $df = 2, 48$, $p < .025$). The mean reaction time in the delayed response condition was 321 msec, while in the immediate response condition it was 463 msec. The session effect reflected an improvement in reaction time over sessions (session 1, 411 msec; session 2, 399 msec; session 3, 366 msec).

The only physiological variables to show differences between the delay conditions were HR MAX-MIN and MIN-BASE scores. These differences can be compared with the differences in reaction time. The smallest amount of deceleration occurred before the GO signal, which initiated faster reactions than the DISCRIMINATIVE-GO signal. Session effects were present in the heart rate BASE and MAX-BASE scores. These two effects are necessarily confounded, but indicated a higher mean heart rate in the third session, which seemed to lead to a small MAX-BASE score. Thus, the fastest reactions occurred during the session with the highest average heart rate. Neither skin conductance nor muscle activity showed any difference due to delay conditions or session.

Heart Rate and Reaction Time—Correlational Analyses. Within-subject correlations between HR and reaction time were computed for each of the six experimental treatments formed by combinations of immediate versus delayed reaction conditions and three shock probabilities. Since within-subject correlations were computationally laborious, initial analyses used only the MAX-MIN scores. These scores were selected because they showed the most consistent differences between experimental treatments. Moreover, the MAX-MIN scores were closely related to the MIN-BASE scores, with a mean between-subject correlation of −.75 across the six treatment combinations, and a range of −.93 to −.59. (A negative correlation means that larger MAX-MIN scores were associated with larger MIN-BASE scores.) Most of the variance in the MAX-MIN scores is thus attributable to cardiac deceleration. Finally, Coquery and Lacey (1966) reported essentially identical correlations between reaction time and HR for MAX-MIN and MIN-BASE type scores.

The within-subject correlations between MAX-MIN scores and reaction time were uniformly low. Using Fisher's Z to average the correlations, the overall mean was found to be −.005. The averages for the six treatment combinations ranged from −.036 to +.017. Further within-subject correlational analyses using MIN-BASE scores did not seem warranted, especially in view of the previously described findings that treatments which produced the least cardiac deceleration and the highest average HR also produced the fastest reactions. However, the between-subject rank correlations were computed using reaction time and MIN-BASE scores, averaged across trials. The overall mean correlation was −.112. (A negative correlation means that faster reactions were associated with greater deceleration.) This overall correlation was not significant, but one individual correlation was. The correlation in the immediate reaction, 0% shock treatment, was −.62 ($p < .01$). Some doubt is raised regarding the meaningfulness of this correlation, however, by the fact that in this same condition the within-subject correlation between MAX-MIN scores and reaction time was only .008. (The correlation between MAX-MIN and MIN-BASE scores for this condition was −.84.) That is, the relationship between cardiac deceleration and reaction time observed across subjects did not hold within subjects across trials.

EMG and Reaction Time. A series of t tests was used to evaluate the relationship between muscle activity and reaction time. The plan of the analysis follows exactly that described in the section on the coupling between somatic and cardiac activity. Table 3 presents the results. The values shown are the mean reaction speed in msec of the first EMG pattern in the left column paired with that for the second. The values are given separately for the immediate and delayed reaction conditions, but are averaged over the shock probability levels. Preliminary analysis showed the direction of the results to be consistent across the shock treatments.

The main findings of Table 3 are contained in the first four rows. These make the same contrasts between EMG patterns as the corresponding rows in Table 2. The results are quite consistent. When EMG bursts occurred just prior to the DISCRIMINATIVE-GO signal, reactions were significantly faster. The same

TABLE 3

Mean reaction times on trials associated with different patterns of EMG activity

EMG Activity	Mean RTs (msec)	
	DISCRIMINATIVE-GO	GO
0/1 versus 0/0	403.6 vs 445.0**	301.0 vs 331.9
1/1 versus 1/0	405.1 vs 480.6**	296.4 vs 294.4
0/1 versus 1/0	394.2 vs 447.4	299.5 vs 345.7
1/1 versus 0/0	405.1 vs 482.7**	296.4 vs 305.2
1/0 versus 0/0	475.8 vs 463.9	339.6 vs 324.2
1/1 versus 0/1	388.2 vs 421.7	296.4 vs 244.5

Note.—Probability values are those for the significance of the difference between reaction times associated with the two EMG patterns. The significance was evaluated using t tests for dependent means, with degrees of freedom varying from 4 to 14.

** $p < .05$.

trend is also apparent for reactions to the delayed GO signal. In the latter case, differences were smaller and nonsignificant; it must be remembered, however, that reactions in general were faster to the GO than to the DISCRIMINATIVE-GO signal and hence smaller and less reliable differences would be expected. These results indicate that the patterns of EMG activity which accompanied the fastest reaction times also were associated with the least amount of cardiac deceleration (see Table 2).

In summary, reaction times were longer, and cardiac deceleration greater, to the DISCRIMINATIVE-GO than to the GO signal. Within experimental treatments, however, there was generally no correlation between cardiac activity and reaction time. One exception was the immediate reaction, 0% shock treatment, where a significant between-subject correlation indicated a possible relationship between cardiac deceleration and fast reaction times. On the other hand, when patterns of EMG activity were compared, cardiac deceleration was consistently less and reaction times faster on those trials where an EMG burst immediately preceded the signal to respond.

Supplementary Interpretive Analyses

Thus far, the results have been organized primarily around the experimental manipulations. This has allowed an objective presentation, but the overall meaning of the results may be obscured by the detail. Further integration is therefore necessary. In this section, supplementary analyses are presented which place the data within an interpretative framework.

Cardic Deceleration and Attention. There is nothing in the present results to indicate that perceptual demands, motor demands, noxiousness, or uncertainty have *unique* influences on anticipatory cardiac deceleration; rather, all seem to contribute in a similar fashion to this phenomenon. This convergence suggests that a common intervening variable might be controlling the cardiac changes—namely, attention.

We define attention as the degree to which the organism is focusing on one

TABLE 4

Mean cardiac deceleration associated with experimental treatments[a]

Shock Probability	Mean Cardiac Decelerations (MIN-BASE scores)		
	Immediate Reaction DISCRIMINATIVE-GO	Delayed Reaction	
		DISCRIMINATIVE	GO
33%	−4.54	−3.98	−2.47[b]
100%	−3.88	−3.08	−2.47[b]
0%	−3.56	−2.76	−2.47[b]

[a] Arranged in order of decreasing attentional demands from left to right and top to bottom.

[b] Shock probabilities combined.

event to the exclusion of others. Berlyne (1960) has presented evidence for four factors leading to heightened attention: complexity, uncertainty, novelty, and conflict. Novelty was not a factor in the present experiment, but the variables of complexity, conflict, and uncertainty were. Thus, the DISCRIMINATIVE-GO, DISCRIMINATIVE, and GO signals varied in complexity (and/or uncertainty[1]) as determined by the number of actual stimulus lights (3 for the DISCRIMINATIVE-GO, 2 for the DISCRIMINATIVE, 1 for the GO). It is also possible to view the anticipation of a motor response as contributing to signal complexity, which would increase still further the attention demands of the DISCRIMINATIVE-GO condition, but decrease the disparity between the DISCRIMINATIVE and GO signals of the delayed reaction group. The 33% shock probability clearly introduced uncertainty into the task. Both complexity and conflict were higher when shock was present (33% and 100% probabilities) than when absent.

Table 4 presents the mean cardiac deceleration (MIN-BASE scores) associated with experimental treatments. Both the rows and columns of Table 4 are arranged in order of decreasing attentional demands. Thus, proceeding from left to right within any row, treatments are arranged from the most attention demanding (DISCRIMINATIVE-GO) to the least (GO). Cardiac deceleration is clearly ranked in terms of attentional demands along this perceptual-motor dimension. The columns of Table 4 indicate the difference in cardiac deceleration as a function of shock treatment. (No shock accompanied the GO signal; therefore, the values in the third column are means of the three shock treatments.) Due to attention caused by uncertainty and conflict, cardiac deceleration should have been greatest with 33% shock, next with 100% shock, and least with 0% shock. Such was the case.

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[1] For this experiment it is not practical to distinguish complexity from uncertainty, except perhaps with reference to the shock probabilities. In general, uncertainty refers to the number of alternative possibilities facing an organism as weighted by the probabilities of these alternatives. Complexity refers to the number and similarity of the psychologically relevant elements. Complexity, although loosely defined, has been shown to have an effect on behavior independent of uncertainty (Berlyne, 1960, Ch. 2).

Although predictions along the rows and columns of Table 4 are rather straightforward, complications arise if one tries to go from the bottom of one column to the top of the next. For example, it is not clear whether the DISCRIMINATIVE-GO, 0% shock condition demands more attention than the DISCRIMINATIVE, 33% shock condition. Different components of complexity, uncertainty, and conflict enter into these conditions and make a precise ranking impossible. Therefore, it is not surprising to find greater cardiac deceleration to the DISCRIMINATIVE signal, 33% shock, than to the DISCRIMINATIVE-GO signal in the 0% and 100% shock conditions. Perhaps this also explains why differences between the MIN-BASE scores prior to the DISCRIMINATIVE and GO signals were significant only for the 33% and 100% shock probabilities. The anticipation of a motor response would tend to make the GO signal equal in complexity to the DISCRIMINATIVE signal in the absence of shock.

Cardiac Acceleration and Sympathetic (SNS) Activity. Obrist, Wood, and Perez-Reyes (1965) demonstrated that anticipatory cardiac deceleration is the result of increased vagal activity, not of decreased sympathetic tonus. Certain conditions, such as awaiting exercise or noxious stimulation, may exert dual and relatively independent effects on HR. On the one hand, by increasing attention, they may contribute to cardiac deceleration, a vagal effect. On the other hand, these same conditions may stimulate sympathetic activity (or inhibit vagal tonus) by triggering adaptive mechanisms unrelated to attention. For example, motor demands may initiate preparations for activity, such as muscular vasodilation, increased cardiac output, and other changes similar to those found *during* an actual somatic response. Similarly, noxiousness and uncertainty may involve anticipatory sympathetic-like arousal.

The expected relationships were generally observed between shock treatments, on the one hand, and cardiac and electrodermal activity, on the other. As previously described, the accelerative limb (MAX-BASE scores) of the HR response was reliably greater when shock (both 33% and 100% probabilities) was expected. The difference between 33% and 100% shock also approached statistical significance, which is in agreement with the findings of Niemelä (1969) that uncertain shock tends to produce greater anticipatory acceleration than does certain shock. The anticipation of shock also produced greater electrodermal activity in comparison with the 0% shock treatment. However, SC did not seem to be influenced by the uncertainty of shock, as was HR.

On the basis of statistical tests described earlier, the motor vs perceptual task requirements had no significant effect on changes in HR or SC. Nevertheless, differences were in the expected direction and further analyses seemed warranted. Thus, the mean MAX-BASE scores (shock probabilities combined), prior to the DISCRIMINATIVE and DISCRIMINATIVE-GO signals, were 2.50 and 3.00 bpm, respectively. That is, acceleration was slightly greater when a motor response was imminent. A similar comparison can be made between the DISCRIMINATIVE and GO signals within the delayed reaction group, 0% shock probability. (In the other shock treatments, the accelerative limb of the HR response prior to the GO signal was confounded by the shock accompanying the preceding DISCRIMINATIVE signal.) The respective MAX-BASE scores were

1.92 and 3.00 bpm, again suggesting greater acceleration when a motor response was required. An *F* test of this difference approached statistical significance ($p < .10$); however, the comparison was, in a sense, post hoc and therefore must be viewed with some caution.

The effect of motor requirements on SC was in the same direction as on cardiac acceleration. Thus, changes prior to the DISCRIMINATIVE and DISCRIMINATIVE-GO signals (shock probabilities combined) were .313 and .389 μmhos, respectively. Similarly, SC increases prior to the DISCRIMINATIVE and GO signals (delayed reaction group, 0% probability) were .035 and .287 μmhos, respectively. This latter difference was significant at the .05 level of confidence, although again the test was post hoc.

The influence of motor demands on cardiac activity can also be seen from the comparisons of HR responses associated with different patterns of EMG activity. When an EMG burst occurred during the last 5 sec prior to a signal, anticipatory deceleration tended to be less; when EMG activity was present in the initial half of the anticipation interval, the accelerative limb of the HR response was accentuated. These results, together with those discussed above, lend support to the notion that motor activity—actual or anticipated—is associated with an elevation of cardiac activity.

Discussion

The results of the preceding analyses lead to the following proposition: *Any factor (e.g., complexity, uncertainty, conflict, and/or novelty) which increases the intensity of attention will tend to produce cardiac deceleration; however, if preparation for, or execution of, the task is accompanied by increased metabolic activity (as in exercise) or any other response involving relative sympathetic dominance (as to shock), then cardiac deceleration will be mitigated if not entirely superceded.* This hypothesis applies to HR changes both during and preceding an event. In the latter case, however, sympathetic-like reactions preparatory to increased metabolic activity, as well as anticipatory emotional reactions, may not be sufficiently strong to overcome attentional demands, especially immediately preceding the expected event.

The above hypothesis accounts for the results of the present experiment and is generally consistent with other data reported in the literature (e.g., Kagan & Lewis, 1965; Graham & Clifton, 1966; Wilson & Duerfeldt, 1967; Chase, Graham, & Graham, 1968). Indeed, many authors have noted the role of attention as a factor in cardiac deceleration. No originality is claimed for the present analysis, only—it is hoped—greater specificity with regard to relevant environmental parameters. Moreover, this hypothesis does not explain the functional significance of cardiac deceleration during attention. On the physiological level, two ostensibly competing explanations have been offered for this phenomenon.

Lacey (1967; Lacey & Lacey, 1958) has argued that cardiac deceleration reduces afferent impulses from the baroreceptors to bulbar centers which otherwise exert an inhibitory influence on higher cortical mechanisms. Cardiac deceleration thus facilitates attention or, in Lacey's terms, sensory-motor integration. On the other hand, Obrist et al. (1970a, 1970b) have argued that cardiac deceleration is only one aspect of a general cessation of somatic activity which accompanies attention-like states. That is, cardiac deceleration may result from, but does not itself facilitate attention.

On the basis of both Lacey's and Obrist's hypotheses, an association would be expected between cardiac deceleration and fast reaction times. For Lacey, cardiac deceleration has a direct functional link in the chain of events leading to sensory-motor integration; for Obrist, the relationship is indirect, with cardiac deceleration and fast reactions both resulting from an inhibition of irrelevant somatic activity. The present data raise questions regarding the adequacy (or generality) of these formulations. For example, the experimental condition (delayed GO signal) which elicited the fastest reactions also produced the least anticipatory HR deceleration. Moreover, with one possible exception, there was no correlation within conditions between reaction time and HR. Of course, the exception might be important for it was found in the condition (immediate reaction, 0% shock) which corresponds most closely to tasks used by Lacey in his studies. However, the major difficulty with Lacey's hypothesis is that the limits of applicability have not been clearly specified. At best, the current findings suggest that the feedback mechanisms proposed by Lacey, although supported by considerable physiological evidence (Lacey, 1967), may have very little influence in most situations.

With regard to Obrist's hypothesis, a strong and weak version must be distinguished. The weak version simply states that somatic activity, by increasing metabolic needs, will tend to increase cardiac activity, either directly or through central mediating processes. It would be difficult to disagree with such a suggestion, which is supported by a large body of physiological data, as well as by the results of the present experiment. Obrist, however, has argued for a much stronger hypothesis, namely, that cardiac responses are invariably linked to somatic events, both reflecting a common biological process related to metabolic needs. This implies that HR deceleration and fast reaction times must be accompanied by an inhibition of somatic activity. (For a similarly strong hypothesis linking cardiac activity to motor demands, see Elliott, 1969.)

The results of this experiment lend little support to the strong version of Obrist's hypothesis. Thus, reaction times were faster, not slower, when the signal to respond was preceded by a burst of EMG activity. These results are consistent with findings by Freeman and others that muscle tension often aids in producing fast reactions (Woodworth, 1938, pp. 328 ff.). Indeed, some of Obrist's own data run counter to his hypothesis. For example, in one experiment (Obrist, Webb, & Sutterer, 1969) *S*s were required to tap gently on the arm of the chair as they awaited a painful electric shock, a time when cardiac deceleration was also occurring. It was therefore concluded that deceleration accompanied the suppression of *irrelevant* somatic activity. The difficulty is, of course, to specify *in advance* what is irrelevant.

In short, it seems that the hypotheses of both Obrist and Lacey may contain part of the truth. However, it is difficult to say which part, since neither hypothesis is sufficiently specific on the psychological (in contrast to physiological) level—that is, with regard to the influence of relevant situational parameters.

The attentional hypothesis presented earlier attempts to specify the role of some of these situational parameters.

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